The Resonance of the Tissues as a Factor in the Transmission of the Pulse and in Blood Pressure.

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Systolic blood pressure in man is measured by the pressure indicated on a manometer scale at the point of disappearance and reappearance of the pulse. When the pressure is raised in the armlet of the Riva-Rocci or Hill–Barnard, or their modifications, or in the bag of the pocket sphygmometer (L. Hill), the pulse is supposed to disappear at the moment when the arterial lumen is obliterated, and to reappear when the patency of the channel is re-established. Consequently every effort has been made to secure that the pressure should be transmitted to the arterial wall as far as possible without loss. Accuracy in instrumental readings has been held to be conditional on such perfect transmission of pressure.

Of late years controversy has ranged round the importance of the arterial wall as a factor in blood pressure, especially in diseased conditions of the wall, *e.g.* arteriosclerosis. One of us (L. Hill) with Russell Wells (2) and Martin Flack (3) has shown the importance of the arterial wall in influencing conduction of the pulse, and has ascribed the high readings obtained in the arteries of the leg in cases of aortic regurgitation to a better conduction of the pulse in contracted and more rigid arteries. There remains for us in this paper to demonstrate another factor, hitherto overlooked, in the taking of blood-pressure observations, namely, the influence on the arterial pulse of the resonance of the tissues permeated with arterioles. The pulse is essentially a phenomenon of periodic vibrations, and by the resonance of the tissues we denote the property of the tissues to further the pulse vibrations by synchronous vibrations of like (positive) periodicity.

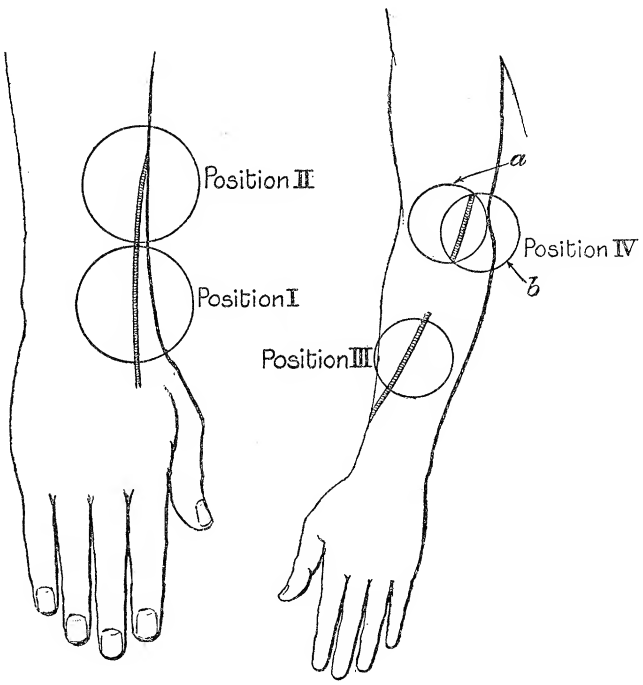
Our observations have been made in the first place on a man, a boiler-maker by trade, aged 53 years, whose arteries show on both arms slight though equal arteriosclerosis. His apex beat is visible within the nipple line, and his cardiac valves are intact. No aneurismal condition is detected.

His right radial artery pursues an aberrant course, curving some 3 inches above the styloid process of the radius over the supinator longus muscle on to the dorsal surface of the forearm, where it runs over the extensor tendons

of the thumb, till it dips between the interossei muscles in the first interosseus space to join the deep carpal arch. The brachial artery in the arm and the radial artery in the forearm are divided by us into certain positions.

Position I (radial artery) denotes the part of the artery on the back of the hand that can be covered by the bag of the L. Hill sphygmometer while space is left for pulse observation distal to it.

Position II (radial artery) denotes that part of the artery which can be



covered by the bag as it curves from the dorsal surface to the palmar surface of the forearm.

Position III (radial artery) denotes the superficial course of the radial artery in the forearm just previous to its dipping deep between the supinator longus muscle and the pronator radii teres.

Position IV (brachial artery) denotes that part of the brachial artery that lies superficially in the antecubital fossa.

In position I the artery lies superficially under the skin, and is placed upon an unyielding bed of bone, the carpal bones, their ligaments and the tendons of extensor muscles. Such an observational site may well be taken as a standard, in the light of which all other positions may be reviewed. The bag of the pocket syhygmometer applied on the artery at position I cannot fail to transmit pressure equally to all parts of the artery

beneath the bag, and there can be no loss of pressure here through the faulty transmission of intervening tissues or through distortion of tissues.

Analysed anatomically position II is similar to position I, while in position III the radial artery courses over the pronatus quadratus and flexor longus pollicis. At position IV the brachial artery lies on the deep tendon of the brachialis anticus muscle.

Taking readings of disappearance and reappearance of the pulse with the pocket sphygmometer we find—

		mm. of Hg.		mm. of Hg.
Position I	pulse disappearance	55	pulse reappearance	50
Position II	„ „	55	„ „	50
Position III	„ „	130	„ „	125
Position IV A	„ „	105	„ „	100
Position IV B	„ „	75	„ „	70

Substituting a bag of water for one of air the readings are—

		mm. of Hg.		mm. of Hg.
Position I	pulse disappearance	55	pulse reappearance	50
Position II	„ „	45	„ „	40
Position III	„ „	130	„ „	125
Position IV A	„ „	119	„ „	109
Position IV B	„ „	65	„ „	63

The subject was in the horizontal position in all cases.

The low reading of 55–50 mm. of Hg at position I cannot be due to any fault in the transmission of pressure through the bag to the arterial wall Consequently, we assume that the pulse has disappeared at 55–50 mm. of Hg before the blood flow has ceased through the artery. In other words, the phenomenon of arresting the pulse by occlusion of the artery is not brought into play in this observation.

Two methods suggest themselves by means of which it can be proved that when the pulse ceases to be felt at position I the arterial flow is still maintained, that the pulse has, as it were, been skimmed off the current.

Keeping the bag of the sphygmometer pressed on position II with a pressure of 180 mm. of Hg, one can strip the blood out of the artery, and, to prevent recurrent flow, fix the artery below, as it dips through the interosseous space. By releasing the pressure at II, the lumen of the empty artery can be felt to fill with blood when the pressure in the bag registers 115–120 mm. of Hg. It can be felt standing out as a bulging cord at 90 mm. of Hg, while the pulse returns at 60 mm. of Hg.

It is possible to place the armlet so as to cover position III and part of

II, while the bag is pressed on part of II and part of I, the following reading can then be taken :—

	mm. of Hg.
Pressure in armlet	90
Pulse disappears below bag at	55

Here there can be no question that the blood flow passes through the pressure of 90 mm. of Hg, and therefore cannot be arrested by a pressure of a bag at 55 mm. of Hg. A further possibility suggests itself that the pulse may be diverted through pressure on the bag, and seek an easier channel through some branch of the radial artery. Against this supposition we suggest: first, that the branch chosen must be a big one, otherwise what the pulse gains in an easier path is lost in the friction due to the narrower lumen; second, that a pulse would never pass back from the bag at 50 mm. of Hg, under the armlet at 90 mm. of Hg. Consequently we conclude that with the bag in position I the pulse is damped down under the bag, while there is but a trifling obstruction to the blood flow in the artery. The blood in the artery below the bag takes on the characters of a venous flow.

The aberrant radial artery where it lies in part of position I, in position II, and position III was covered by the armlet, and while preventing the recurrent ulnar pulsation, a reading was taken. The pulse was then found to disappear and reappear between the limits of 120–130 mm. of Hg. Consequently the aberrant radial artery in positions I and II, overlying bone ligaments and tendons, can withstand a pressure of, say, 110 mm. of Hg without the pulse being damped down. But with the bag of the pocket sphygmometer at position I or at position II, the pulse is removed from the blood current with a pressure of 55–60 mm. of Hg. Yet, according to physical laws, the pressure is equally delivered to the elastic wall of the artery by both instruments. The problem is seen then to depend on the air contained in the armlet in the one case, and on the air contained in the bag in the other. It is not a matter solely of pressure in the air of the armlet or of the bag, but the important factor is the state of the air in both cases as regards periodic vibrations.

The air in the armlet is in a state of periodic vibration. These vibrations depend on the pulsation of the mass of tissues which surround the ulna and radius and are embraced by the armlet. At every beat of the heart the incompressible blood is pumped into the tissues through arteries large and small, and the pulse of each and every artery is directed as much outwards into the tissues as inwards upon the blood stream. Consequently the tissues become a pulsating mass, as can be

registered on a plethysmograph curve. When the bag of the pocket sphgymometer is applied to the artery, either at position I or at position II, the pulsations in the air of the bag are at a minimum, because the tissues lying under the bag are comparatively pulseless. In the case of the armlet, with its wider embrace of pulsing tissues, the air shows pulsations more or less synchronous to the pulse in the artery, the arterial pulse is thereby strengthened and enabled to resist the damping-down effect of the armlet. Consequently the pressure applied to the arterial wall may be increased from 60 to 100 or 110 mm. of Hg, as the case may be, and yet the pulse persists, provided the medium through which the pressure is applied is itself in a condition of like periodic vibration. Of course, the vibrations must be of such a period as will strengthen the pulse of the artery and not oppose it.

It is on this fundamental experiment that the hypothesis of the resonance of the tissues is grounded. By this hypothesis we can explain the various readings obtained by the same instrument (*e.g.* bag of pocket syhygmometer) at positions I, II, III and IV. Position II is obviously similar to position I.

In position III the radial artery lies as we trace it centrally, first on the pronator quadratus, and then on the flexor longus pollicis. When the bag is applied to the artery in position III, there are beneath it fleshy tissues with numerous arteries in them. Consequently, the tissues below the bag are throbbing more or less synchronously with the pulse in the radial artery at position III. The air of the bag is then in a state of periodic vibration, as in the case of the air of the armlet. Accordingly, the reading becomes the high one of 130–135 mm. of Hg. The damping-down effect of the bag on the pulse has been compensated for by the resonance of the tissues beneath it. Readings with the bag of the pocket sphygmometer placed at position IV have been noted to vary from 60 to 100 mm. of Hg. In taking these readings, the recurrent ulnar pulsation can be damped down at position I, and the pulse felt at position II. Such variable readings do not occur haphazardly; it can be demonstrated that they depend on the varying anatomical condition of the areas below the bag. Such areas may be classified into areas of high resonance and areas of low resonance.

If a diagonal line is drawn through the centre of the superficial brachial artery at position IV (see Diagram), the bag of the sphygmometer can be so placed that 1/3 of the bag lies to the radial side of the artery and 2/3 on the ulnar side, or, the same length of artery being covered as before by the bag, 2/3 of the bag can lie to the radial side of the artery and 1/3 to the ulnar side.

These positions are indicated by the circle (*a*) and the circle (*b*).

It is to be noted that the same length of artery is under pressure in both cases. The bags are covered with the hand in a precisely similar manner, yet the pulse at *a* reappears at 90 mm.; at *b*, reappears at 60 mm.

This difference can be explained by an analysis of the tissues underlying the bag in either position. In position *a*, 2/3 of the bag lies on the fleshy belly of the supinator longus and biceps, and over the arterial anastomosis of the radial recurrent artery and the superior profunda artery. In position *b*, 2/3 of the bag lies on the tendinous insertions of the flexor group of muscles. Here the arterial supply is much less. Consequently, the resonance of the tissues in position *a* is greater than the resonance of the tissues in position *b*, and the pulse suffers a great damping-down in position *b*.

Here we have no question of loss of pressure through overlying or distorting tissues. The tissues over the artery are the same in both cases. The pressure on each point of the circumference of the bag is the same. Consequently, it must be that, in position *a*, the air delivering the pressure is in a state of greater periodic vibration than the air in the bag in position *b*. The vibrations that underlie the phenomenon of sound are transmitted in water as in air. We find that when water is substituted in the bag for air the same results are obtained. The water takes on the periodic vibrations of the resonating tissues.

L. Hill and Russell Wells (2) have recently shown how important a factor in the pulse curve is the lability of the arterial wall. It has also been shown by L. Hill and Martin Flack (3) that, when an artery is freed from the tissues, and thereby deprived of the support of the tissues round its wall, the pulse curve is much affected. The lability of the wall is called into play, and the systolic pressure of the heart is spent in distending the wall of the artery. It was possible, then, that the artery lying more or less superficially at positions II, III, and IV, would have its wall distended, so that the pulse arriving under the bag at position I would be already damped down before pressure was applied to the artery at position I.

Our experiments show that, at position I, with a pressure, say, of 60 mm. of Hg, the pulse is skimmed off the blood current, but the arterial flow remains. Consequently, the block on the blood flow is not an absolute one.

Experiments were made by supporting the superficial artery with the armlet and with the bag of another sphygmometer, to determine whether such support played any part in the production of the low pressure reading at position I.

Our results show that no matter what pressure is raised in the armlet on

positions III and part of II, the pressure in the sphygmometer bag covering part of position II and part of position I required to obliterate the pulse remains the same. On the other hand, when the artery is, in addition, supported by varying pressures at position IV, the reading becomes 5–15 mm. higher. Simultaneous support in positions IV, III, and part of II, makes the reading at part of position II and part of position I higher by 5–10 mm. of Hg.

Table where Supporting Pressure is Applied successively at Elbow and Forearm.

Supporting pressure of sphygmometer bag at elbow, position IV.	Supporting pressure in armlet over part of III and part of II.	Sphygmometer bag at part of I and part of II.
mm. of Hg.	mm. of Hg.	mm. of Hg.
Experiment I ... 40	40	75 disappearance of pulse.
0	0	65 ,, ,,
Experiment II ... 40	40	73 ,, ,,
0	0	63 ,, ,,
Experiment III ... 40	40	65 ,, ,,
0	0	50 ,, ,,
Experiment IV ... 0	0	55 reappearance ,,
0	40	55 ,, ,,
40	40	65 ,, ,,
Experiment V ... 0	0	55 ,, ,,
0	40	55 ,, ,,
40	40	65 ,, ,,

Table where Supporting Pressure is Applied at Forearm only.

Supporting pressure of armlet in position III and part of II.	Sphygmometer bag on part of I and part of II.
mm. of Hg.	mm. of Hg.
0	50 reappearance of pulse.
10	50 ,, ,,
20	50 ,, ,,
30	50 ,, ,,
40	50 ,, ,,
50	50 ,, ,,
60	50 ,, ,,
70	50 ,, ,,
80	50 ,, ,,
90	50 ,, ,,
100	50 ,, ,,
110	50 ,, ,,

Note.—It is important to commence from zero and work upwards and not raise the pressure to 110 mm. of Hg all at once, because venous congestion, which is rapidly accommodated for when rising from zero, otherwise proves a disturbing factor.

Table where Supporting Pressure is Applied as far as possible Simultaneously at Elbow and Forearm.

Supporting pressure of armlet applied at position III and part of II.	Supporting pressure of sphygmometer bag at position IV.	Sphygmometer bag at part of position I and part of II.
		Increase in mm. of Hg on previous reading before application of supporting pressure at elbow and forearm.
mm. of Hg.	mm.	
Experiment I ... 20	20	5–10
Experiment II ... 30	30	5–10
Experiment III ... 40	40	5–10
Experiment IV ... 50	50	5–10
	Note.—Care must be taken that the bag at IV is applied as in Diagram III *a*.	

Note.—It is not possible to apply these pressures at Positions IV, III and part of II with perfect synchronism, as the pressure cannot be raised in the armlet to 50 mm. without two compressions of the pump.

Accordingly, provided one guards against errors from change in the arterial wall through manipulation—we have noted that after many readings with the bag at position I the artery becomes obviously harder and the reading rises—and, provided one constantly guards against a rise in arterial pressure during an experiment, then lack of support of the wall may account for a loss of pressure of 5–10–15 mm. of Hg. But such lack is obviously unable to account for the low reading at position I of 50–60 mm. of Hg. Further, the experimental observations with the sphygmometer bag on varying positions at IV show that the main factor must be the resonance of the tissues.

But low readings with the sphygmometer bag are not confined to aberrant radial arteries. Thus one may observe the same phenomenon on the dorsalis pedis artery.

Reading with bag on dorsalis pedis horizontal position—

	mm. Hg.		mm. Hg.
Dorsalis pulse disappears	85.	Right radial pulse disappears	145.
„ „ reappears	80	„ „ „ reappears	140.

In this case the dorsalis pedis available was short and the foot was fleshy.

In another case where the dorsalis pedis is longer and the tissues surrounding it scantier, then—

	mm. Hg.		mm. Hg.
Dorsalis pulse disappears at	55.	Left radial pulse disappears at	135.
„ „ reappears „	50	„ „ „ reappears „	130.

Subject in the horizontal posture.

In yet another case the pulse disappeared at 35–40 mm. Hg.

The anterior tibial artery in the leg is overlapped in the upper part of the leg by the tibialis anticus muscle, in the lower part of the leg by the extensor longus digitorum, extensor proprius hallucis, and anterior annular ligament. The dorsalis pedis artery is overlapped by the anterior annular ligament and by the innermost tendon of the extensor brevis digitorum. Consequently, the artery above the point of application of the sphygmometer bag is well supported. Yet the readings are similar to readings on positions I and II of the aberrant radial artery.

The low blood-pressure readings obtained with Hill's pocket sphygmometer on the aberrant radial artery, or on the dorsalis pedis artery, are due to the absence of the resonance of the tissues. Provided one could, in the forearm, tie every artery except the radial, and every large branch of the radial artery, one would find then that the blood-pressure readings taken by Hill's pocket sphygmometer, or by the armlet method, would approximate closely to the low readings found in the aberrant radial artery.

Another method of demonstrating the effect of resonance on the pulse is the following:—Blood-pressure readings are taken in an individual in the upright position, from the forearm held at the level of the heart. The systolic blood-pressure is found to be 120 mm. of Hg (disappearing pulse index). A similar reading is found in the other arm. One arm is then fully extended above the head, and the forearm, from the tips of the fingers to the elbow, is bandaged tightly to render the limb ischæmic. An armlet is fitted to the upper arm, and the pressure is raised in it well above the systolic pressure to prevent the blood flowing into the ischæmic limb. The bandage is then removed, and the arm lowered to the heart level. Hill's pocket sphygmometer is now placed on the forearm covering the same position as before (the position is previously outlined with ink) and the radial artery is blocked with one finger to prevent a pulse from the ulnar recurrent artery; the pressure in the armlet is then let down rapidly by pulling the tube off the metal connection of the compressing bulb. When the first pulses are felt at the wrist, the bag of the sphygmometer is pressed on to the artery until the pulse is damped down, a pressure of 70 mm. of Hg suffices to do this. Soon the pulse reappears below the bag, and the bag has to be pressed on with, say, a

pressure of 80 mm. of Hg before the pulse again disappears. We find the systolic blood-pressure readings rise successively from 70 mm. to 80–90–100–110–120–130–140 mm. of Hg. There may, or may not, be a rebound effect when the blood pressure rises, for a short period, above what it was at the commencement of the experiment, and above the reading in the forearm of the other arm.

The ischæmic limb on the abolition of pressure in the armlet on the upper arm is found to gradually swell and becomes red. There is obviously a marked vaso-dilatation.

Bayliss (4) has shown that when the blood pressure is taken off a limb or an organ, *e.g.* by blocking the abdominal aorta, an increase in volume of the limb or organ occurs when the block of the aorta is removed. Bayliss offers no proof as to which part of the vascular mechanism dilates in this reaction. A study of the phenomenon in a limb with an aberrant radial artery during this experiment gives a clue to the vascular conditions present in the reaction. The aberrant radial artery can be seen to dilate. It stands out like a small worm on the back of the wrist. The veins on the forearm also dilate. It is unlikely that the arterioles are constricted when there is visible an increased blush of the capillary area. We conclude that during Bayliss' phenomenon, after a bandage has been used to make the limb ischæmic, the main arteries as well as the arterioles of the limb dilate.

This can be proved by tracings taken with the Dudgeon sphygmograph from the aberrant radial artery on position I. We use weight extension to fix the Dudgeon. The base line of the tracings is seen to progressively rise as the artery dilates. Care must be taken to fix the limb effectively during this experiment.

A further proof that the main arteries are dilated can be got by plunging the congested limb into ice-cold water. After a period in the cold water the artery is felt to be very much constricted, and this is confirmed by visual examination. Massage of the artery brings it back to its original dilated condition.

A modification of this experiment, viz. releasing the artery and taking the blood pressure in the ischæmic limb as the limb fills with blood, can be performed. The ischæmic limb with an armlet on the upper arm at a pressure well above the ascertained systolic blood pressure can be plunged into ice-cold water with ice in it. After a short period the limb, withdrawn from the ice-cold water, is found to be thoroughly chilled and is dried by mopping lightly without rubbing. Rubbing might dilate the arteries. When the pressure is let down suddenly in the armlet, at first the pulse can be damped down by 40 to 50 mm. of Hg, then the pressure rises, but much more

slowly, to normal or above normal. This is the important point, that the blood-pressure reading in the radial artery rises much more slowly in the cold ischæmic limb wherein the arteries are constricted than in the warm ischæmic limb wherein the arteries are dilated. At the conclusion of the experiment, when the blood pressure is back to normal, the aberrant radial artery still feels like a whipcord—highly contracted. Massage of the whipcord artery will bring it back to the worm-like condition which obtained in the congested limb.

Accordingly we can conclude that the phenomenon occurs in the dilated or in the contracted artery—it is immaterial which. Consequently the initial low blood pressures (as measured by the disappearance of pulse) on allowing the blood to enter the arteries are independent of the state of the arterial wall. They are also independent of the peripheral resistance.

Blood-pressure estimations were made on the aberrant radial artery at the close of these experiments on the warm limb (now congested) and on the cold limb.

When the systolic pressure had arisen to the normal 120–130 mm. of Hg in the forearm in the warm limb the reading obtained at position I on the dilated aberrant radial artery was 50–60 mm. of Hg. In the cold limb when the blood-pressure reading in the forearm was found to be 150–160 mm. of Hg (the same as the initial pressure in the individual tested), the constricted aberrant radial artery gave a reading of 70–80 mm. of Hg.

We conclude, therefore, that the pulse in either the dilated artery or the contracted artery can be damped down by a pressure 70–80 mm. Hg or so below normal. Experiments similar to the above, and with like result, can be performed on the dorsalis pedis artery.

We have traced in the ischæmic limb the rise in the size of the beat of the radial artery, or of the dorsalis pedis artery, or of the aberrant radial artery (at position I) using both Mackenzie's polygraph and the weight-extension method and the Dudgeon sphygmograph, and blocking the artery below to prevent the recurrent pulse. When the armlet is compressed in the upper arm and the pressure suddenly let go, one notes that the beat in the congested limb returns quicker to its normal size than in the ischæmic limb. In all cases the beat takes longer to come to normal when the weight-extension Dudgeon is used than when the tracings are taken by the polygraph. The weight-extension method of applying the Dudgeon avoids the plethysmographic effect of the polygraph (Lewis). One often finds the pulse takes a minute to return to its maximal swing, *i.e.* until the surrounding tissues are filled with blood and resonate with it.

It might be argued in the light of the fact that the return of the maximal

beat is slower in the ischæmic limb than in the congested limb, that we have herein a natural explanation of the initial low pressure readings. The pulse beats in the ischæmic limb are of feeble force, consequently the bag of the sphygmometer applied to the artery naturally damps down the feeble beats. But we have shown that maximal beats, whether the artery is dilated or contracted, suffer a damping-down in the aberrant radial artery at extremely low blood pressures. Thus in one experiment, when the systolic blood pressure was taken at heart level by Hill's sphygmometer in the forearm at position III and was found to be 120–130 mm. of Hg, the returning pulse in the ischæmic limb at position III was damped down at 70 mm., and when the blood pressure rose at position III to 120–130 mm. the pulse in the aberrant radial artery at the back of the wrist at position I where maximal beats could be recorded was damped down at 50–60 mm. The feebler pulse beats in the forearm on the radial artery at position III required 70–80 mm. to damp them down. The maximal beats on the same radial artery at position I required only 50–60 mm. of Hg to damp them down. We see, in fact, that the pulse beat, no matter how forcible, can be damped down by a pressure 70 mm. of Hg or so below normal blood pressure.

It might be argued that the low blood-pressure readings obtained in this experiment represent the actual blood pressure in the radial artery, that there has been a fall of head of pressure as the blood flows into the ischæmic limb. It is not probable that the head of blood pressure would fall greatly, because the blood flows through the narrow arterioles and still narrower capillary bed. No matter whether the arterioles and capillary bed are full or empty, the resistance to the blood stream remains in the friction of the vessel walls. But blocking the radial artery below the point of measurement effectively removes the objection that there is a fall of head of pressure. It might be argued that the fall of pressure continues down the ulnar artery. But by blocking the radial artery one converts the radial artery into a side tube measuring lateral pressure from the brachial at the elbow, and the lateral pressure of the brachial artery at the elbow would not fall. Further, one can block both radial and ulnar arteries, and the pressure readings taken from the forearm of the ischæmic limb show the same progressive rise. We conclude that on suddenly lowering the pressure in the armlet the blood pressure rapidly becomes normal, and the low blood-pressure readings, as measured by the disappearance of the pulse, are false, both in the ischæmic limb and in the cold ischæmic limb.

The explanation of these low blood-pressure readings lies in the diminished resonance of the empty tissues. The mass of tissue below the bag is not

tense with blood and does not vibrate strongly with the pulse, consequently the sphygmometer bag acts as a damper. The rise of the pulse to maximal is aided by the resonance of the tissues. But whether the pulse beat is maximal or not it is bound to suffer damping down so long as the resonance of the surrounding tissues is feeble.

It will be noted that the blood pressure in the cold ischæmic limb returns much more slowly to normal than in the warm ischæmic limb. Here the arterioles of the limb are contracted; consequently the blood takes longer to percolate into the ischæmic tissues, the drum-head takes longer to tighten up, and the resonating effect consequently longer to develop. After dilatation has been produced in the vessels of a limb, repeatedly made ischæmic, it is less easy to obtain the staircase effect. The bandaging has then to be done very tightly; on letting go the brachial artery the blood rushes in swiftly, the skin blushes, and the maximal beat quickly returns.

Many years ago Hürthle (5) noted that the diastolic pressures taken simultaneously with a manometer at the femoral artery and at the carotid artery were nearly similar, while the systolic pressure at the femoral exceeded that of the carotid by roughly 68 mm. of Hg. Dawson (6) corroborates this statement, working with the maximum and minimum manometer, but points out the diastolic pressure in the femoral is always slightly lower than the diastolic in the carotid.

We would advance the explanation of the higher systolic and lower diastolic readings in terms of the resonance theory. The abdomen functionates as a resonator of the pulse, because each organ in it—liver, spleen, kidney, intestines, etc.—are all pulsating and the cavity is a closed one. Descent of the diaphragm is compensated for by an outward movement of the abdominal wall. The abdominal wall is an elastic structure. Consequently the systolic pulse in the aorta and great vessels is surrounded by more or less synchronous pulsations, which, like the well adjusted tap on the moving pendulum, augment its swing.

In the case of the higher blood-pressure readings in the leg arteries, compared to the arm readings found by Hill, Flack, Holtzman and Rowlands (1) in cases of aortic disease, we believe the same resonating effect of the abdominal cavity is at work, together with the better conduction of the pulse wave down the tighter abdominal and leg arteries.

It was suggested by one of us (L. Hill) in 'Further Advances in Physiology' (7), that the kidney functionated largely through the mechanism of the arterial pulse. "In the case of the kidney the blood in the capillary network, the tissue lymph, and the urine in the tubules are all at one and the same pressure—the capillary-venous pressure. The whole kidney is

expanded by each arterial pulse, and drops of urine may be squeezed thereby into the pelvis from the mouths of the tubules." Recent work by R. A. Gesell (8) has shown that the excretion of the urine, the chlorides, urea and nitrogen is dependent on the arterial pulse. It is to enable the pulse to be driven to the capillary areas in the kidney or other organ that the mechanism of a resonation of the tissues is called for. Without some such mechanism the pulse would be inevitably damped down, especially during the varying abdominal pressures found with deep inspiration, forced expiration, defæcation, etc.

Further, we would advance the view that by abdominal resonance the pulse wave is assisted to the most distant peripheral regions of the body. The aortic pulse finds its way to the tips of the fingers in aortic disease and to the toes. The longer path is compensated for by abdominal resonance.

Resonation of the tissues must be held to play an important part in the transmission of the pulse, and thereby to save the work of the heart. The work of the heart we know is largely conserved by the elastic recoil of the arteries. But this elastic recoil of the arteries is aided by the resonance of the tissues. Every artery is in intimate relationship with its immediate neighbour. The pulse of one individual artery is aided by the pulses of the other arteries. The vigour of the circulation depends on the tone of the tissues, on the tautness of skin and muscle, and particularly of the abdominal wall. The hardened body of the trained athlete swings in full resonance with the pulse of his heart; the soft, flabby, ill-conditioned body of the sedentary worker offers a poor slack drum for his heart to thump.

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